

Product datasheet for **KN213623RB**

NLRP3 Human Gene Knockout Kit (CRISPR)

Product data:

Product Type:	Knockout Kits (CRISPR)
Format:	2 gRNA vectors, 1 RFP-BSD donor, 1 scramble control
Donor DNA:	RFP-BSD
Symbol:	NLRP3
Locus ID:	114548
Components:	KN213623G1 , NLRP3 gRNA vector 1 in pCas-Guide CRISPR vector (GE100002) KN213623G2 , NLRP3 gRNA vector 2 in pCas-Guide CRISPR vector (GE100002) KN213623RBD , donor DNA containing left and right homologous arms and RFP-BSD functional cassette. GE100003 , scramble sequence in pCas-Guide vector

Disclaimer: These products are manufactured and supplied by OriGene under license from ERS. The kit is designed based on the best knowledge of CRISPR technology. The system has been functionally validated for knocking-in the cassette downstream the native promoter. The efficiency of the knock-out varies due to the nature of the biology and the complexity of the experimental process.

RefSeq: [NM_001079821](#), [NM_001127461](#), [NM_001127462](#), [NM_001243133](#), [NM_004895](#), [NM_183395](#)

UniProt ID: [Q96P20](#)

Synonyms: AGTAVPRL; All; AVP; C1orf7; CIAS1; CLR1.1; FCAS; FCAS1; FCU; MWS; NALP3; PYPAF1



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Summary:

This gene encodes a pyrin-like protein containing a pyrin domain, a nucleotide-binding site (NBS) domain, and a leucine-rich repeat (LRR) motif. This protein interacts with the apoptosis-associated speck-like protein PYCARD/ASC, which contains a caspase recruitment domain, and is a member of the NLRP3 inflammasome complex. This complex functions as an upstream activator of NF-kappaB signaling, and it plays a role in the regulation of inflammation, the immune response, and apoptosis. The SARS-CoV 3a protein, a transmembrane pore-forming viroporin, has been shown to activate the NLRP3 inflammasome via the formation of ion channels in macrophages. Mutations in this gene are associated with familial cold autoinflammatory syndrome (FCAS), Muckle-Wells syndrome (MWS), chronic infantile neurological cutaneous and articular (CINCA) syndrome, neonatal-onset multisystem inflammatory disease (NOMID), keratoendotheliitis fugax hereditaria, and deafness, autosomal dominant 34, with or without inflammation. Multiple alternatively spliced transcript variants encoding distinct isoforms have been identified for this gene. Alternative 5' UTR structures are suggested by available data; however, insufficient evidence is available to determine if all of the represented 5' UTR splice patterns are biologically valid. [provided by RefSeq, Aug 2020]

Product images:
